CASE REPORT

Atraumatic splenic rupture: a cause of haemorrhagic shock secondary to low-molecular-weight heparin

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Abstract

Anticoagulant treatment can be a risk factor for splenic rupture and the use of low-molecular-weight heparin is becoming more frequent for diverse indications. We report a case of an 86-year-old man admitted in the intensive care unit because of haemorrhagic shock due to atraumatic splenic rupture. He was on anticoagulant treatment. An abdominal ultrasound was suggestive of spontaneous splenic rupture which was confirmed by an abdominal computed tomography scan. The patient died before a laparotomy was performed. Spontaneous splenic rupture, not related to trauma, is uncommon. It is associated with many causes such as cancer, pregnancy or anticoagulant treatment. A bedside ultrasound can be very useful in assessment of unstable patients.

Introduction

Splenic rupture is a potentially life-threatening complication usually occurring after blunt abdominal trauma. The atraumatic rupture of the spleen is very rare, approximately 0.1-0.5% in the literature. However, there are many diseases or underlying conditions which can cause, or increase the risk of, splenic rupture.[1-7] These include obesity, infectious diseases such as mononucleosis or malaria, haematological or rheumatoid diseases, severe pre-eclampsia, cancer by metastasis or direct spleen invasion, splenic infarction, splenic artery aneurysm, pancreatitis, amyloidosis, and medical procedures such as colonoscopy and drugs (tinzaparin and enoxaparin). No aetiology is described in 5-7% of cases of atraumatic splenic rupture. The signs of atraumatic splenic rupture are abdominal pain, left shoulder pain and, more rarely, shock. These signs are the same as traumatic rupture, but the difference is the history of trauma. An abdominal CT scan is the most valued tool used to diagnose splenic rupture, but bedside ultrasound using the focused abdominal sonography trauma (FAST) exam has been described as an effective method in non-trauma patients who need immediate resuscitation.[2]

Case report

An 86-year-old man was admitted to hospital to investigate a left pleural effusion with dyspnoea and loss of weight (4 kg) for one month. The patient had no fever, no cough, and no chest pain or palpitations. The patient was only on acenocoumarol because of atrial fibrillation. He had no other past medical history. After reversing the anticoagulation, a pleural effusion puncture was performed and a chest tube was inserted. Analysis of the pleural fluid obtained by thoracentesis indicated an exudative type. Microbiological samples and a biopsy of pleural effusion were sent off to the lab. Anticoagulation was started again. The dose of enoxaparin used was 1 mg by 1 kg of the patient's weight (65 kg). Each 60 mg/12 hour dose was administered subcutaneously. The patient had received four doses when he deteriorated suddenly: he was unresponsive, his respirations were agonal, his pulse was weak and thready and his blood pressure was unobtainable. Intravenous fluids and catecholamine pressors were started, the patient was intubated rapidly and transferred to the intensive care unit (ICU). Upon admission to the ICU, the patient's blood pressure was 60/40 mmHg, pulse 150 bpm and temperature 36.8°C. Physical examination revealed a diffusely tender and distended abdomen, with signs of peritoneal irritation. The respiratory examination indicated decreased breath sounds in the lower left hemithorax, with dullness on percussion. Laboratory results revealed the following values: white blood cell count 12×10^3/µl, haemoglobin 6.5 g/dl (previously 13 g/dl), platelet count 74×10^3/µl, creatinine 3.03 mg/dl (previous creatinine 0.8 mg/dl), sodium 143 mEq/l, potassium 5.6 mEq/l, lactate 20 mmol/l, INR 1.76; cephalin time 35.5 sec, prothrombin activity 49.8%, fibrinogen 88 mg/dl, blood arterial gas pH 7.07, pCO_2 34.6 mmHg, pO_2 97.5 mmHg, and bicarbonate 10 mmol/l. Due to the sudden onset and signs of hypovolaemic shock, there was a high clinical suspicion of gastrointestinal bleeding or rupture of an aortic aneurysm. There was no evidence of external bleeding and the patient's family denied any past or recent abdominal

trauma. A pleural effusion and correct positioning of the chest tube was observed on the chest X-ray and in the lung ultrasound. No pneumothorax was seen. A bedside ultrasound following the FAST protocol was performed showing no pericardial effusion, free fluid in the abdomen and in Morison's pouch with an anechoic image around the liver, free fluid in the splenorenal recess and around the spleen with hyperechoic images highly suggestive of parenchymal injury, pleural effusion in the left lung without any damage in the bladder or aorta. Intravenous saline, blood products (packed red blood cells, fresh frozen plasma, platelets, and fibrinogen), and escalating doses of catecholamine pressors (reaching a maximum of 1.2 µg/kg/min) were administered. A general surgeon was called and asked for a CT scan, which was performed when the patient was stabilised (blood pressure 100/60 mmHg, lactate 11.2 mmol/l, pH 7.20, pCO_2 32 mmHg, pO_2 102 mmHg, bicarbonate 16 mmol/l). CT scan confirmed haemoperitoneum and a large subcapsular splenic haematoma (grade III according to the American Association for the Surgery of Trauma) with active

Figure 1A. Axial contrast-enhanced CT showed portal venous gas (black arrow) and high-attenuation fluid (white arrow) in perihepatic area, the hepatorenal fossa and the lesser sac
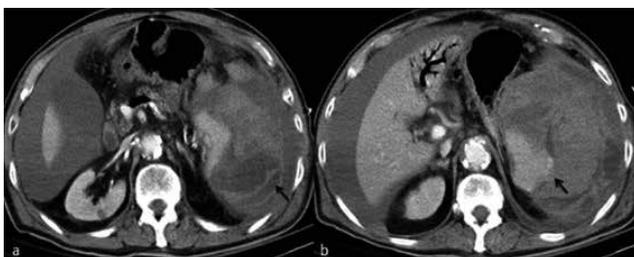
Figure 1B. Portal venous phase axial CT images show (A) subcapsular haematoma (black arrow) and perisplenic blood collection; and (A) active haemorrhage from spleen injury seen as contrast extravasation (black arrow)

bleeding, and signs of extensive acute mesenteric ischaemia (*figures 1A and 1B*). The patient died before a laparotomy was performed, 1.5 hours after ICU admission. The pathology report confirmed rupture of the splenic capsule and revealed a lung adenocarcinoma without any lesions on the spleen. The chest tube had been correctly placed in the thorax.

Discussion

The use of low-molecular-weight heparin (LMWH) is becoming more frequent for diverse indications. Heparins act indirectly by binding to antithrombin, which converts antithrombin from a slow to a rapid inactivator of coagulation factors (e.g., thrombin or factor IIa, factor Xa). LMWH is metabolised in the liver and excreted by the kidney. Renal clearance accounts for approximately 10 to 40%. Individuals with a creatinine clearance <30 ml/min have a significantly increased plasma level (approximately 65%) and generally require dose adjustment.[8]

The major complication of anticoagulant therapy is bleeding. [8,9] Specific risk estimates depend on patient factors (e.g., age, comorbidities, underlying indication for anticoagulation), heparin dose, activated partial thromboplastin time (aPTT), and the use of other antithrombotic therapies (e.g., antiplatelet agents). Bleeding risk is also increased in patients with trauma and/or undergoing invasive procedures. The management of bleeding in a patient receiving heparin depends on the location and severity of bleeding, the thromboembolic risk, and the level of the aPTT (for heparin) or anti-factor Xa activity (for LMWH). The degree of anticoagulation is important both for predicting the course of the bleeding episode and for determining which interventions will be required. The anticoagulation status depends on the specific agent, dose, time since the last dose, and renal (and to a lesser extent hepatic) function. The need for urgent heparin reversal is individualised according to the site and severity of bleeding and the degree of anticoagulation. If urgent reversal is required, heparin is discontinued and protamine sulphate is administered. LMWH has a longer duration of action than unfractionated heparins and it is less easily inactivated with protamine sulphate, making it more difficult to rapidly stop therapy. Other available strategies for reversing the anticoagulant effect include pro-haemostatic therapies such as antifibrinolytic agents and desmopressin and prothrombin complex concentrates (PCCs), which are concentrates of coagulation factors and anticoagulants purified from plasma. They contain high levels of three or four coagulation factors (II, IX, and X in 3-factor PCCs; II, VII, IX, and X in 4-factor PCCs), along with protein C and S.

The management of shock[9] due to severe bleeding may require transfusion of red blood cells. Platelet transfusion is not used to reverse the anticoagulant effect in patients with a normal platelet count, but prevention of coagulopathy with early transfusion of plasma and platelets is critical in patients with severe haemorrhage. Fresh frozen plasma may be given as part of

a massive transfusion protocol, to replace coagulation factors lost by bleeding and replacement of packed red blood cells, which do not contain coagulation factors at replacement levels. There is no evidence to support the use of fresh frozen plasma as a reversal strategy in direct oral anticoagulant-associated bleeding.

Some studies have found that anticoagulant treatment can be a risk factor for splenic rupture. Blankenship et al. reported on a series of 17 patients with splenic haemorrhage who were on anticoagulant or thrombolytic therapy.[10] Kocael et al.[11] reviewed 12 patients with splenic rupture and 33% were receiving anticoagulant-antiaggregant treatment. Several mechanisms underlying atraumatic splenic rupture have been described in literature:[1,5,7] thinning of the capsule and the creation of a mass effect making the spleen more fragile and thus more vulnerable to trauma (minor traumatic impacts could cause splenic injury in patients with splenomegaly). In patients on anticoagulant treatment, any trivial traumatism may cause parenchymal fissures and secondary bleeding, aggravated by anticoagulation. Deprez et al. described a spontaneous splenic rupture following embolic infarction and anticoagulant treatment.[7] Thrombotic occlusion of the splenic arterial tree leads to infarction, which can be partial or complete, single or multiple. Use of anticoagulation is related to haemorrhagic transformation of previously infarcted areas. One of the causes of infarction is embolisation from atrial fibrillation. Our patient had atrial fibrillation, but there was no evidence of splenic infarction. Neither were there any findings of systemic infection, primary tumour, metastasis, or abscess in the spleen. A case of splenic rupture after a left-sided thoracotomy has been described.[12] In our case a chest tube was placed correctly in the patient's left side the day before and it had not damaged the spleen. Correct positioning of the chest tube was confirmed by imaging techniques and in the autopsy. Therefore, the rupture of the spleen seems to be associated with the anticoagulant treatment. The doses received were adjusted by renal function (a normal measure for adjustment) and the patient's weight. A case of spontaneous splenic and liver rupture has been reported in a patient on warfarin, even when it was administered in the therapeutic range.[4]

The mortality caused by atraumatic splenic rupture is 12%, but it is increased up to 20-30% in ruptures associated with anticoagulant treatment. Splenomegaly, the presence of underlying neoplastic diseases and age >40 years appear to be important predictors of mortality.[1,10] Our patient was 86 years old. However, one of the most important causes of mortality is delay in the diagnosis. The clinical state of the patient and the 20 mmol/l lactate suggested a severe and evolved scenario. This case illustrates that it is very important to suspect atraumatic splenic rupture in patients with predisposing factors and to utilise bedside ultrasound in the assessment of critically ill patients, especially in the event of shock.[3] In our case, ultrasound was

preformed which indicated free intraperitoneal fluid with mixed echogenicity of the spleen, suggestive of splenic injury. Those findings should have been enough to perform an urgent laparotomy. However, patient stabilisation after treatment allowed for confirmation of the ultrasound findings with a CT scan. Even though CT confirmation was done very quickly (20 minutes), it was not as fast as performing an ultrasound. Ultrasound has limitations including clinician experience and acoustic window, but we think systematic ultrasound evaluation should be used to make a differential diagnosis in shock. The finding of extensive acute mesenteric ischaemia (confirmed in pathology examination) was secondary to hypovolaemic shock and it implies a high mortality. Emergency splenectomy is a life-saving intervention in cases of splenic ruptures with haemodynamic instability.

This case is interesting in several aspects. On one hand, anticoagulant drugs are increasingly used in medical practice. Complications are more likely in the elderly, and in those with chronic conditions. Although splenic rupture is more often related to trauma, physicians must be aware of some conditions that can lead to atraumatic rupture. On the other hand, the prompt recognition of shock can be the most important life-saving measure. Point-of-care ultrasound can help avoid delays in diagnosis in patients with shock.

Disclosures

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